What Is Claimed Is:

1. A method of treating a pathological condition characterized at least partially by involvement of the NK-1 receptor, said method comprising,

administering to a mammal in need thereof, a therapeutically effective amount of at least one oligonucleotide or oligonucleotide analog which interferes with the function or production of NK-1 receptors.

2. The method of Claim 1, wherein said interference with said function or production of said NK-1 receptors involves at least one nucleic acid in the NK-1 receptor pathway.

3. The method of Claim 2, wherein said nucleic acid is one or more selected from the group consisting of DNA, RNA, tRNA, mRNA and rRNA.

4. The method of Claim 1, wherein said oligonucleotide comprises RNA in the form of at least one ribozyme.

5. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is one or more selected from oligonucleotides and oligonucleotide analogs that are complementary to nucleic acid in said NK-1 receptor pathway.

6. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of DNA antisense oligonucleotides and oligonucleotide analogs, RNA antisense oligonucleotides and oligonucleotide analogs, DNA sense oligonucleotides and oligonucleotide analogs, RNA sense oligonucleotides and oligonucleotide analogs, aptamers and ribozymes.

7. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is at least one selected from those that are complementary to at least a portion of the NK-1 receptor DNA or RNA shown in SEQ. ID Nos. 2, 4, 6 and 8.

8. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of those shown in SEQ. ID Nos. 9-59.

9. The method of Claim 1, wherein said mammal is a human.

10. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is applied by intrathecal infusion to the spinal canal.

11. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

12. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 20 to 25 nanomoles per kilogram of body weight of said mammal.

13. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 15 to 300 nanomoles per kilogram of body weight of said mammal.

14. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 50 to 600 micrograms per kilogram of body weight of said mammal.

15. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 200 to 400 micrograms per kilogram of body weight of said mammal.

16. The method of Claim 1, wherein the amount of said oligonucleotide or oligonucleotide analog that is administered is from 250 to 350 micrograms per kilogram of body weight of said mammal.

17. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is administered via intravenous infusion.

18. The method of Claim 1, wherein said oligonucleotide or oligonucleotide analog is administered by one or more routes selected from the group consisting of oral, parenteral, rectal, sub-cutaneous, mucosal, buccal, transdermal, intravaginal, nasal, nasal inhalation, pulmonary inhalation, iontophoresis through the skin, iontophoresis through mucosal or buccal membranes, dermal patch, epidural, intracranial, intrapharyngeal, sublingual, intra-articular, intramuscular, and subcutaneous.

19. The method of Claim 1, wherein said pathological condition is one or more selected from the group consisting of dermatological disorders, immune disorders, autoimmune disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, neuropathic disorders, central aspects of chronic or acute pain,

peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

20. The method of Claim 19, wherein said vascular disorder is migraine.

21. The method of Claim 19, wherein said nervous system disorder is at least one selected from the group consisting of anxiety, psychosis, and schizophrenia.

22. A pharmaceutical preparation comprising at least one oligonucleotide or oligonucleotide analog selected from the group consisting of oligonucleotides and oligonucleotide analogs that interfere with the function or production of at least a portion of said NK-1 receptor.

23. The pharmaceutical preparation of Claim 22, in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

24. The pharmaceutical preparation of Claim 22, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of those shown in SEQ. ID Nos. 9-59.

25. The pharmaceutical preparation of Claim 22, wherein said oligonucleotide or oligonucleotide analog is complementary to any nucleic acid in said NK-1 receptor pathway.

26. The pharmaceutical preparation of Claim 25, wherein said nucleic acid in said NK-1 receptor pathway is RNA.

27. The pharmaceutical preparation of Claim 22, wherein said nucleic acid in said NK-1 receptor pathway is DNA.

28. The pharmaceutical preparation of Claim 22, wherein said preparation is administered to treat one or more pathological conditions selected from the group consisting of dermatological disorders, immune disorders, autoimmune disorders, neuropathic disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, central aspects of chronic or acute pain, peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

29. The pharmaceutical preparation of Claim 26, wherein said vascular disorder is migraine.

30. The pharmaceutical preparation of Claim 26, wherein said nervous system disorder is at least one selected from the group consisting of anxiety, psychosis, and schizophrenia.

31. A kit for treating or diagnosing a pathological condition, said kit comprising a pharmaceutical preparation comprising at least one oligonucleotide or oligonucleotide analog selected from the group consisting of oligonucleotides and oligonucleotide analogs that interfere with the function or production of at least a portion of said NK-1 receptor, and instructions for administering said pharmaceutical preparation to a mammal.

32. The kit of Claim 31, wherein said pathological condition is one or more selected from the group consisting of dermatological disorders, immune disorders, autoimmune disorders, neuropathic disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, central aspects of chronic or acute pain, peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

33. The kit of Claim 31, wherein said at least one oligonucleotide or oligonucleotide analog is in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

34. A method of treating, attenuating or preventing pain comprising, administering to a mammal in need thereof, a therapeutically effective amount of at least one compound that interferes with the function or production of NK-1 receptors.

35. The method of Claim 34, wherein said compound is at least one selected from the group consisting of oligonucleotides or oligonucleotide analogs, and non-nucleotide disruptor compounds.

36. The method of Claim 34, wherein said interference with the function or production of said NK-1 receptors involves at least one nucleic acid in the NK-1 receptor pathway.

37. The method of Claim 36, wherein said nucleic acid is one or more selected from the group consisting of DNA, RNA, tRNA, mRNA and rRNA.

38. The method of Claim 35, wherein said oligonucleotide comprises RNA in the form of at least one ribozyme.

39. The method of Claim 35, wherein said oligonucleotide or oligonucleotide analog is one or more selected from oligonucleotide or oligonucleotide analogs that are complementary to nucleic acid in said NK-1 receptor pathway.

40. The method of Claim 35, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of DNA antisense oligonucleotides or oligonucleotide analogs, RNA antisense oligonucleotides or oligonucleotide analogs, DNA sense oligonucleotides or oligonucleotide analogs, RNA sense oligonucleotides or oligonucleotide analogs, aptamers and ribozymes.

41. The method of Claim 35, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of those shown in SEQ. ID. Nos. 9-59.

42. The method of Claim 34, wherein said mammal is a human.

43. The method of Claim 35, wherein said disruptor is one or more selected from the group consisting of methylation compounds, de-methylation compounds, antibodies to nucleic acids, mutagens, intercalation compounds, gyrases, ligases, and methylases.

44. The method of Claim 34, wherein said compound is applied by intrathecal infusion to the spinal canal.

45. The method of Claim 34, wherein said compound is administered by one or more routes selected from the group consisting of oral, buccal, mucosal, parenteral, rectal, sub-cutaneous, transdermal, intravaginal, nasal, nasal inhalation, pulmonary inhalation, iontophoresis through the skin, iontophoresis through mucosal or buccal membranes, dermal patch, epidural, intracranial, intrapharyngeal, sublingual, intra-articular, intramuscular, and subcutaneous.

46. The method of Claim 34, wherein the amount of said compound that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

47. The method of Claim 34, wherein the amount of said compound that is administered is from 20 to 25 nanomoles per kilogram of body weight of said mammal.

48. The method of Claim 34, wherein the amount of said compound that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

49. The method of Claim 34, wherein the amount of said compound that is administered is from 50 to 600 micrograms per kilogram of body weight of said mammal.

50. The method of Claim 34, wherein the amount of said compound that is administered is from 200 to 400 micrograms per kilogram of body weight of said mammal.

51. The method of Claim 34, wherein the amount of said compound that is administered is from 250 to 350 micrograms per kilogram of body weight of said mammal.

52. The method of Claim 34, wherein said compound is administered via intravenous infusion.

53. The method of Claim 34, wherein said pain is characterized as peripheral pain, chronic pain, acute pain, neuropathic pain, pain relating to psychiatric disorders, and pain relating to central nervous system disorders.

54. The method of Claim 34, wherein said pain is chronic pain.

55. The method of Claim 34, wherein said pain is neuropathic pain.

56. The method of Claim 34, wherein said pain is characterized by at least one from the group consisting of hyperalgesia, allodynia, neuralgia, and dysesthesia.

57. The method of Claim 35, wherein said non-nucleotide disruptor acts directly upon nucleic acid in said NK-1 pathway.

58. The method of Claim 34, wherein said non-nucleotide disruptor does not act directly upon nucleic acid in said NK-1 pathway.

59. A pharmaceutical preparation useful for preventing, attenuating or treating pain, comprising at least one compound selected from the group consisting of compounds that interfere with the function or production of NK-1 receptors.

60. The pharmaceutical preparation of Claim 59, wherein said compound is at least one selected from the group consisting of oligonucleotides or oligonucleotide analogs, and non-nucleotide disruptor compounds.

61. The pharmaceutical preparation of Claim 59, in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

62. The pharmaceutical preparation of Claim 59, wherein said oligonucleotide or oligonucleotide analog is complementary to any nucleic acid in said NK-1 receptor pathway.

63. The pharmaceutical preparation of Claim 59, wherein said oligonucleotide or oligonucleotide analog is at least one selected from those that are complementary to at least a portion of the NK-1 receptor DNA or RNA of those shown in SEQ. ID Nos. 2, 4, 6 and 8.

64. The pharmaceutical of Claim 59, wherein said disruptor is one or more selected from the group consisting of methylation compounds, de-methylation compounds, antibodies to nucleic acids, mutagens, intercalation compounds, gyrases, ligases, and methylases.

65. A kit for treating, diagnosing, attenuating or preventing pain, said kit comprising a pharmaceutical preparation comprising at least one compound selected from the group consisting of oligonucleotides and oligonucleotide analogs, and non-nucleotide disruptors that interfere with the function or production of at least a portion of the NK-1 receptor, and instructions for administering said compound to a mammal.

66. The kit of Claim 65, wherein said pain is characterized as peripheral pain, chronic pain, acute pain neuropathic pain, pain relating to psychiatric disorders, and pain relating to central nervous system disorders.

67. The kit of Claim 65, wherein said pain is characterized by at least one from the group consisting of hyperalgesia, allodynia, neuralgia, and dysesthesia.

68. The kit of Claim 65, wherein said compound is in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

69. A method of treating, attenuating or preventing an inflammatory condition characterized at least partially by activation of the NK-1 receptor comprising, administering to a mammal in need thereof, a therapeutically effective amount of at least one compound that interferes with the function or production of NK-1 receptors.

70. The method of Claim 69, wherein said compound is at least one selected from the group consisting of oligonucleotides or oligonucleotide analogs and non-nucleotide disruptor compounds.

71. The method of Claim 69, wherein said interference with the function or production of said NK-1 receptors involves at least one nucleic acid in the NK-1 receptor pathway.

72. The method of Claim 69, wherein said nucleic acid is one or more selected from the group consisting of DNA, RNA, tRNA, mRNA and rRNA.

73. The method of Claim 69, wherein said oligonucleotide comprises RNA in the form of at least one ribozyme.

74. The method of Claim 70, wherein said oligonucleotide or oligonucleotide analog is one or more selected from oligonucleotide or oligonucleotide analogs that are complementary to nucleic acid in said NK-1 receptor pathway.

75. The method of Claim 70, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of DNA antisense oligonucleotide or oligonucleotide analogs, RNA antisense oligonucleotide or oligonucleotide analogs, DNA sense oligonucleotide or oligonucleotide analogs, RNA sense oligonucleotide or oligonucleotide analogs, aptamers and ribozymes.

76. The method of Claim 70, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of those shown in SEQ. ID Nos. 9-59.

77. The method of Claim 69, wherein said mammal is a human.

78. The method of Claim 70, wherein said oligonucleotide or oligonucleotide analog is applied by intrathecal infusion to the spinal canal.

79. The method of Claim 69, wherein said compound is administered by one or more routes selected from the group consisting of oral, mucosal, buccal, parenteral, rectal, sub-cutaneous, transdermal, intravaginal, nasal, nasal inhalation, pulmonary inhalation, iontophoresis through the skin, iontophoresis through mucosal or buccal membranes, dermal patch, epidural, intracranial, intrapharyngeal, sublingual, intra-articular, intramuscular, and subcutaneous.

80. The method of Claim 69, wherein the amount of said compound that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

81. The method of Claim 69, wherein the amount of said compound that is administered is from 20 to 25 nanomoles per kilogram of body weight of said mammal.

82. The method of Claim 69, wherein the amount of said compound that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

83. The method of Claim 69, wherein the amount of said compound that is administered is from 50 to 600 micrograms per kilogram of body weight of said mammal.

84. The method of Claim 69, wherein the amount of said compound that is administered is from 200 to 400 micrograms per kilogram of body weight of said mammal.

85. The method of Claim 69, wherein the amount of said compound that is administered is from 250 to 350 micrograms per kilogram of body weight of said mammal.

86. The method of Claim 69, wherein said compound is administered via intravenous infusion.

87. The method of Claim 69, wherein said inflammation is characterized as peripheral inflammation, chronic inflammation, acute inflammation, inflammation relating to psychiatric disorders, and inflammation relating to central nervous system disorders.

88. The method of Claim 69, wherein said inflammation is chronic inflammation.

89. The method of Claim 69, wherein said inflammation is neuropathic inflammation.

90. The method of Claim 69, wherein said inflammation is characterized by at least one from the group consisting of hyperalgesia, allodynia, neuralgia, and dysesthesia.

91. The method of Claim 70, wherein said non-nucleotide disruptor is one or more selected from the group consisting of methylation compounds, de-methylation compounds, antibodies to nucleic acids, mutagens, intercalation compounds, gyrases, ligases, and methylases.

92. The method of Claim 70, wherein said non-nucleotide disruptor acts directly upon nucleic acid in said NK-1 pathway.

93. The method of Claim 70, wherein said non-nucleotide disruptor does not act directly upon nucleic acid in said NK-1 pathway.

94. A pharmaceutical preparation useful for treating inflammation, comprising at least one compound selected from the group consisting of compounds that interfere with the function or production of NK-1 receptors.

95. The pharmaceutical preparation of Claim 78, wherein said compound is at least one selected from the group consisting of oligonucleotides or oligonucleotide analogs and non-nucleotide disruptor compounds.

96. The pharmaceutical preparation of Claim 78, in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

97. The pharmaceutical preparation of Claim 78, wherein said oligonucleotide or oligonucleotide analog is one or more selected from the group consisting of those shown in SEQ. ID Nos. 9-59.

98. The pharmaceutical preparation of Claim 78, wherein said oligonucleotide or oligonucleotide analog is complementary to any nucleic acid in said NK-1 receptor pathway.

99. The pharmaceutical preparation of Claim 78, wherein said oligonucleotide or oligonucleotide analog is at least one selected from those that are complementary to at least a portion of the NK-1 receptor DNA or RNA of those shown in SEQ. ID Nos. 2, 4, 6 and 8.

100. A kit for treating, diagnosing, attenuating or preventing inflammation, said kit comprising a pharmaceutical preparation comprising at least one compound selected from the group consisting of oligonucleotides and oligonucleotide analogs, and non-nucleotide disruptors that interfere with the function or production of at least a portion of the NK-1 receptor, and instructions for administering said compound to a mammal.

101. The kit of Claim 100, wherein said inflammation is characterized as peripheral inflammation, chronic inflammation, acute inflammation, dermatological inflammation, neuropathic inflammation, inflammation relating to psychiatric disorders, and inflammation relating to central nervous system disorders.

102. The kit of Claim 100, wherein said inflammation is characterized by at least one from the group consisting of hyperalgesia, allodynia, neuralgia, and dysesthesia.

103. The kit of Claim 100, wherein said compound is in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

104. The kit of Claim 100, wherein said compound is a is one or more disruptors selected from the group consisting of methylation compounds, de-methylation compounds, antibodies, mutagens, intercalation compounds, gyrases, ligases, and methylases

105. A method of treating a pathological condition characterized at least partially by involvement of the NK-1 receptor, said method comprising,

administering to a mammal in need thereof, a therapeutically effective amount of at least one disruptor that interferes with the function or production of NK-1 receptors.

106. The method of Claim 105, wherein said interference with said function or production of said NK-1 receptors involves at least one nucleic acid in the NK-1 receptor pathway.

107. The method of Claim 105, wherein said disruptor is not a nucleic acid or nucleic acid analog.

108. The method of Claim 105, wherein said disruptor is one or more selected from the group consisting of methylation compounds, de-methylation compounds, antibodies to nucleic acids, mutagens, intercalation compounds, gyrases, ligases, and methylases.

109. The method of Claim 105, wherein said NK-1 disruptor acts directly upon nucleic acid in said NK-1 pathway.

110. The method of Claim 105, wherein said NK-1 disruptor does not act directly upon nucleic acid in said NK-1 pathway.

111. The method of Claim 106, wherein said nucleic acid is one or more selected from the group consisting of DNA, RNA, tRNA, mRNA and rRNA.

112. The method of Claim 111, wherein said RNA is at least one ribozyme.

113. The method of Claim 105, wherein said mammal is a human.

114. The method of Claim 105, wherein said disruptor is applied by intrathecal infusion to the spinal canal.

115. The method of Claim 105, wherein said disruptor is administered via intravenous infusion.

116. The method of Claim 105, wherein said disruptor is administered by one or more routes selected from the group consisting of oral, parenteral, rectal, sub-cutaneous, transdermal, intravaginal, nasal, nasal inhalation, pulmonary inhalation, iontophoresis through the skin, iontophoresis through mucosal or buccal membranes, dermal patch, epidural, intracranial, intrapharyngeal, sublingual, intra-articular, intramuscular, and subcutaneous.

117. The method of Claim 105, wherein the amount of said disruptor that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

118. The method of Claim 105, wherein the amount of said disruptor that is administered is from 20 to 25 nanomoles per kilogram of body weight of said mammal.

119. The method of Claim 105, wherein the amount of said disruptor that is administered is from 15 to 30 nanomoles per kilogram of body weight of said mammal.

120. The method of Claim 105, wherein the amount of said disruptor that is administered is from 50 to 600 micrograms per kilogram of body weight of said mammal.

121. The method of Claim 105, wherein the amount of said disruptor that is administered is from 200 to 400 micrograms per kilogram of body weight of said mammal.

122. The method of Claim 105, wherein the amount of said disruptor that is administered is from 250 to 350 micrograms per kilogram of body weight of said mammal.

123. The method of Claim 105, wherein said pathological condition is one or more selected from the group consisting of dermatological disorders, autoimmune disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, central aspects of chronic or acute pain, peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

124. The method of Claim 105, wherein said vascular disorder is migraine.

125. The method of Claim 105, wherein said nervous system disorder is at least one selected from the group consisting of anxiety, psychosis, and schizophrenia.

126. A pharmaceutical preparation comprising at least one disruptor selected from the group consisting of compounds that interfere with the function or production of at least a portion of an NK-1 receptor.

127. The pharmaceutical preparation of Claim 126, in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.

128. The pharmaceutical preparation of Claim 126, wherein said disruptor acts upon RNA in said NK-1 receptor pathway.

129. The pharmaceutical preparation of Claim 126, wherein said disruptor acts upon DNA in said NK-1 receptor pathway.

130. The pharmaceutical preparation of Claim 126, wherein said disruptor acts upon at least one protein in said NK-1 receptor pathway.

131. The pharmaceutical preparation of Claim 126, wherein said preparation is administered to treat one or more pathological conditions selected from the group consisting of dermatological disorders, autoimmune disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, central aspects of chronic or acute pain, peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

132. The pharmaceutical preparation of Claim 126, wherein said vascular disorder is migraine.

133. The pharmaceutical preparation of Claim 126, wherein said nervous system disorder is at least one selected from the group consisting of anxiety, psychosis, and schizophrenia.

134. A kit for treating or diagnosing a pathological condition, said kit comprising a pharmaceutical preparation comprising at least one disruptor selected from the group of compounds that interfere with the function or production of at least a portion of said NK-1 receptor, and instructions for administering said disruptor to a mammal.

135. The kit of Claim 134, wherein said disruptor is one or more selected from the group consisting of methylation compounds, de-methylation compounds, antibodies to nucleic acids, mutagens, intercalation compounds, gyrases, ligases, and methylases.

136. The kit of Claim 134, wherein said pathological condition is one or more selected from the group consisting of dermatological disorders, autoimmune disorders, cardiovascular disorders, vascular disorders, gut inflammation, arthritis, airway disorders, central aspects of chronic or acute pain, peripheral aspects of chronic or acute pain, psychiatric disorders, and central nervous system disorders.

137. The kit of Claim 134, wherein said disruptor is in admixture with at least one pharmaceutically acceptable substance from the group consisting of excipients, penetration enhancers, stabilizers, absorption enhancers and carrier compounds.